1: Int Angiol 1997 Dec;16(4):250-4 Related Articles, Books, LinkOut

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Treatment of severe Raynaud's syndrome by injection of autologous blood pretreated by heating, ozonation and exposure to ultraviolet light (H-O-U) therapy.

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Related Resources

OBJECTIVE: To determine the effect of re-injection of small samples of autologous blood, pretreated with heat, ozone and ultraviolet light (H-O-U therapy) in patients with severe Raynaud's syndrome. EXPERIMENTAL DESIGN: Open trial in 4 patients. SETTING: Temperature/humidity controlled vascular laboratory. PATIENTS: Severe Raynaud's syndrome of more than 5 years duration and defined as more than 5 attacks daily or 10 attacks in one week, at least half of which were painful and lasting for more than 30 minutes. Three patients were refractory to infusions of Iloprost. INTERVENTIONS: Patients were treated daily or on alternate days for a two to three weeks period by re-injection of citrated autologous blood pre-treated with heat, ozone and ultraviolet light (H-O-U therapy). MEASURES: Clinical observations; mean equilibrated hand temperature (infrared thermography); distributive and microcirculatory blood-flow (venous occlusion strain-gauge plethysmography, infrared photoplethysmography, laser Doppler flowmetry) iontophoresis of acetylcholine and sodium nitroprusside; estimations: serum levels of 6-keto-PGF1alpha and serum levels of anti-hsp65 antibody. RESULTS: Reduction or abolition of Raynaud's attacks for at least three months after treatment. Mean equilibrated hand temperature increased but did not normalise. Blood flow parameters improved but did not reach statistical significance. Iontophoresis of acetylcholine showed an increase in laser Doppler flowmetry which was statistically significant. Serum levels of 6-keto-PGF1alpha, fell significantly in three patients. Serum levels of anti-hsp65 antibody fell in the one patient which was followed sequentially. CONCLUSIONS: H-O-U therapy may prove useful in patients with severe Raynaud's syndrome.

Publication Types:
- Clinical trial

PMID: 9543222 [PubMed - indexed for MEDLINE]

1: J Cutan Med Surg 2000 Jul;4(3):132-7 Related Articles, Books

NEW

PubMed Services

The effect of VAS972 on allergic contact hypersensitivity.

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Related Resources

BACKGROUND: Contact hypersensitivity (CHS) is a Th1-mediated immune response that can be down-regulated by immunosuppressive agents such as cyclosporine and environmental stimuli such as ultraviolet light. Recently, an immunomodulation therapy, VAS972, has been developed which is believed to down-regulate the Th1 arm of the immune response. This VAS972 involves modifying autologous blood by controlled exposure to the oxidizing agent ozone and UVC light, at an elevated temperature ex vivo. The processed blood is then administered by intramuscular injection. OBJECTIVE: To further evaluate the immune modulating effect of VAS972. METHODS: We examined the effect of VAS972 treatment on CHS. Contact hypersensitivity was induced with dinitrofluorobenzene (DNFB) in animals receiving VAS972- processed blood, control blood, or saline. A preliminary study was also conducted to evaluate the effect of plasma and cellular fractions of processed blood. RESULTS: Mice injected with VAS972-processed blood demonstrated a significantly lower (46%) CHS response than controls. Histologic examination of challenged ear skin from control mice displayed edema with a significant lymphocytic infiltration, whereas animals administered processed blood demonstrated a reduction in lymphocytic infiltration. Mice injected with either plasma or the cellular fraction of the VAS972-treated blood also demonstrated a significant suppression (49% and 41%, respectively). CONCLUSION: The results of this study demonstrated that VAS972 suppresses CHS and cellular infiltration. Furthermore, the plasma and cellular components of the VAS972 treatment were also able to induce immunosuppression. This further supports the hypothesis that VAS972 down-regulates the Th1 arm of the immune response.

PMID: 11003717 [PubMed - indexed for MEDLINE]

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1: J Immunol 2000 Sep 1;165(5):2390-7 Related Articles, Books, LinkOut

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Full text article at www.jimmunol.org

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Stress renders T cell blasts sensitive to killing by activated syngeneic NK cells.

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Related Resources

Exposure of primary T cell blasts to stress in the forms of heat, hydrogen peroxide, or high-density growth conditions resulted in a state of enhanced susceptibility to killing by syngeneic IL-2-activated NK cells or lymphokine-activated killer cells, but not to killing by CTL. Cytotoxicity was perforin mediated and was not due to decreased target expression of total MHC class I. The levels of stress used had little effect on cell viability. For thermal stress, sensitization increased with temperature, required a minimum exposure time, and disappeared when cells were given a long enough recovery time. Our data support a model that predicts that activated NK cells play a role in the immunosurveillance of nontransformed stressed cells in normal animals.

PMID: 10946262 [PubMed - indexed for MEDLINE]

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